

*Suffolk County
Vector Control &
Wetlands
Management
Long Term Plan &
Environmental
Impact Statement*



**Task 3: Literature Search
Book 2 Part 3: Susceptibility of Other Organisms
to West Nile Virus**

Prepared for:

**Suffolk County Department of Public Works
Suffolk County Department of Health Services
Suffolk County, New York**

CASHIN ASSOCIATES, P.C.
1200 Veterans Memorial Highway, Hauppauge, NY

January 2005

**SUFFOLK COUNTY VECTOR CONTROL AND WETLANDS MANAGEMENT
LONG - TERM PLAN AND ENVIRONMENTAL IMPACT STATEMENT**

PROJECT SPONSOR

Steve Levy
Suffolk County Executive



Department of Public Works

Charles J. Bartha, P.E.
Commissioner
Richard LaValle, P.E.
Chief Deputy
Leslie A. Mitchel
Deputy Commissioner

Department of Health Services

Brian L. Harper, M.D., M.P.H.
Commissioner
Vito Minei, P.E.
Director, Division of Environmental Quality

PROJECT MANAGEMENT

Project Manager: Walter Dawydiak, P.E., J.D.
Chief Engineer, Division of Environmental Quality, Suffolk County Department of Health Services

Suffolk County Department of Public Works, Division of Vector Control

Dominick V. Ninivaggi
Superintendent
Tom Iwanejko
Entomologist
Mary E. Dempsey
Biologist

Suffolk County Department of Health Services, Office of Ecology

Martin Trent
Acting Chief
Kim Shaw
Bureau Supervisor
Robert M. Waters
Bureau Supervisor
Laura Bavaro
Senior Environmental Analyst
Erin Duffy
Environmental Analyst
Phil DeBlasi
Environmental Analyst
Jeanine Schlosser
Principal Clerk

| SUFFOLK COUNTY LONG TERM PLAN CONSULTANT TEAM | |
|---|---|
| Cashin Associates, P.C. | Hauppauge, NY |
| Subconsultants | |
| Cameron Engineering, L.L.P. | Syosset, NY |
| Integral Consulting | Annapolis, MD |
| Bowne Management Systems, Inc. | Mineola, NY |
| Kamazima Lwiza, PhD | Stony Brook University, Stony Brook, NY |
| Ducks Unlimited | Stony Brook, NY |
| Steven Goodbred, PhD & Laboratory | Stony Brook University, Stony Brook, NY |
| RTP Environmental | Westbury, NY |
| Sinnreich, Safar & Kosakoff | Central Islip, NY |
| Bruce Brownawell, PhD & Laboratory | Stony Brook University, Stony Brook, NY |
| Anne McElroy, PhD & Laboratory | Stony Brook University, Stony Brook, NY |
| Andrew Spielman, PhD | Harvard School of Public Health, Boston, MA |
| Richard Pollack, PhD | Harvard School of Public Health, Boston, MA |
| Wayne Crans, PhD | Rutgers University, New Brunswick, NJ |
| Susan Teitelbaum, PhD | Mount Sinai School of Medicine, NY |
| Zawicki Vector Management Consultants | Freehold, NJ |
| Michael Bottini, Turtle Researcher | East Hampton, NY |
| Robert Turner, PhD & Laboratory | Southampton College, NY |
| Christopher Gobler, PhD & Laboratory | Southampton College, NY |
| Jerome Goddard, PhD | Mississippi Department of Health, Jackson, MS |
| Sergio Sanudo, PhD & Laboratory | Stony Brook University, Stony Brook, NY |
| Suffolk County Department of Health Services, Division of Environmental Quality | Hauppauge, NY |

Primary research for this report was conducted by Cashin Associates (personnel including Joel Banslaben, Brian Parker, and David J. Tonjes, PhD). It was edited and revised in response to comments by Cashin Associates (personnel including David Tonjes, PhD). Review was provided Suffolk County Department of Public Works, Division of Vector Control, and Suffolk County Department of Health Services (personnel including Erin Duffy and Phil de Blasi). Additional comments have been received from _____.

TABLE OF CONTENTS

| | |
|--|-----------|
| List of Abbreviations | iv |
| EXECUTIVE SUMMARY | 1 |
| 1. INTRODUCTION..... | 4 |
| 2. EFFECTS OF WNV ON NON-HUMAN SPECIES..... | 6 |
| 2.1 Birds..... | 7 |
| 2.1.1 Corvids..... | 8 |
| 2.1.2 Raptors | 8 |
| 2.1.3 Songbirds | 9 |
| 2.1.4 Storks | 9 |
| 2.1.5 Sage-Grouse..... | 10 |
| 2.1.6 Domesticated Birds..... | 10 |
| 2.2 Mammals..... | 11 |
| 2.2.1 Horses | 12 |
| 2.2.2 Household Pets..... | 12 |
| 2.2.3 Rodents | 13 |
| 2.2.4 Non-Human Primates..... | 13 |
| 2.2.5 Other Mammals | 14 |
| 2.3 Reptiles | 15 |
| 3. POTENTIAL ECOLOGICAL IMPACT | 16 |
| 3.1 Impacts on Endangered Species..... | 16 |
| 3.2 Ecological Niches | 17 |
| 4. POTENTIAL IMPACT ON HUMANS..... | 18 |
| 4.1 West Nile Incidence..... | 18 |
| 4.2 Transmission via Farm Animals | 18 |
| 4.3 Consumption of Infected Meat | 18 |
| 5. INDIRECT IMPACTS | 20 |
| 5.1 Loss of Natural Controls of Diseases/Pests | 20 |
| 5.2 Economic Impact | 20 |
| REFERENCES..... | 22 |

List of Acronyms and Abbreviations

| | |
|--------|---|
| CDC | Centers for Disease Control and Prevention |
| NYSDEC | New York State Department of Environmental Conservation |
| USEPA | United States Environmental Protection Agency |
| USGS | United States Geological Survey |
| US | United States |
| WNV | West Nile Virus |

Executive Summary

West Nile Virus (WNV) was first observed in North America in 1999, when multiple cases were recorded in New York City. Since that time, nearly 10,000 human infections have been confirmed and over 250 deaths have resulted from the epidemic. In addition to impacts on humans, the disease has adversely affected animals.

WNV is a disease that is spread primarily by mosquito vectors, which infect host species by biting them while in search of nutrient-rich blood. Over 40 species of mosquitoes have been identified as WNV vectors in the United States, most notably the *Culex* species, which are known for breeding in standing water polluted with organic material. The most commonly identified hosts for the virus are horses and avian species, such as corvids. Birds are considered reservoir hosts for their ability to re-infect mosquitoes with the virus. Horses, and most mammals, are labeled dead-end hosts due to their inability to pass the disease onto other mosquitoes because of low levels of the virus in the bloodstream of the host.

The impacts of WNV on animal species and their habitats are important due to the inextricable links between vector and hosts. Many of the ecological impacts of WNV are not well understood, due to its recent introduction to the North American continent. It appears that birds play the largest role in the vector-host relationship, acting both as a reservoir for the disease and as a means for geographic dispersion. American crows are often seen as the sentinel species for WNV monitoring in the US, as most epidemics have been accompanied by observations of significant corvid mortality. In addition, ecologists have surmised that the rapid dispersion of WNV in North America is connected to seasonal bird migrations.

While the total number of fatalities among animal species is unknown, it is likely that the totals reach into the hundreds of thousands. Avian species, most notably corvids such as crows, have been subject to significant effects from the disease. For example, over 4,000 cases of West Nile infection were confirmed for American crows in New York State in 2000. The kinds of birds infected to date include:

- raptors
- songbirds

- geese
- greater sage-grouse
- domestic chickens and turkeys.

Mammals have also experienced significant impacts from WNV. The greatest effects have been observed in horses where hundreds to thousands of mortalities occur each year. Fortunately, most mammal species are considered dead-end hosts and do not re-infect mosquitoes with the virus. Several cases of non-human primate infections have been recorded in the US, all in captive settings. In most cases, the non-human primate did not show clinical illness from West Nile infection. As of 2004, 29 mammalian species had been infected including:

- horses
- chipmunks
- skunks
- squirrels
- wolves
- sheep
- goats
- bats
- seals
- monkeys
- domestic cats and dogs.

Another group of animals potentially impacted by WNV is reptiles. Outbreaks of WNV have been recorded in both alligators and crocodiles. All reptilian outbreaks have occurred in captive

settings, although it is suggested that wild reptiles could be a potential reservoir for the disease. Studies have shown that the transmission of WNV, in some cases, may be due to ingestion of infected horsemeat that is used as feed at commercial alligator and crocodile farms.

In addition to the direct impacts on animal species, overall ecological and indirect impacts of WNV may result from the disease. Ecological niches most likely affected by the disease include those that occur near areas of high mosquito activity, such as coastal and wetland habitats. In addition, urban wildlife may be especially susceptible to outbreaks of the virus due to habitat overlap with *Culex* spp., the suspected prime mosquito vectors. However, observations leading to these conclusions may be an artifact of monitoring effort than actual differences in occurrence. Another potential ecological impact of WNV is its effects on threatened or geographically limited species. Significant impacts of the disease on local populations could lead to critical species levels.

Due to the connections between WNV, mosquitoes, and human and non-human hosts, it is of the utmost importance to understand the ecology of the virus when making public health decisions. In addition, the impacts of mosquito control cannot be completely assessed, except in the context of the human and ecological effects of mosquito-borne diseases. This portion of the literature search, therefore, reports on the effects of WNV on non-human vertebrate species, and some of the indirect impacts on both human and non-human species that may result.

1. Introduction

WNV was first isolated from a woman in the West Nile district of Uganda in 1937. Originally endemic to Africa, western Asia and the Middle East, the virus was soon found in the Eastern Hemisphere, expanding throughout parts of Europe and Australia during the latter half of the 21st century. The first recorded incidences of WNV in the US occurred during the summer of 1999 when the Flushing Medical Center in Queens admitted three patients with a variety of neurological illnesses (Marra et al., 2004). By the end of 1999, WNV had led to 62 confirmed infections and seven deaths in the New York City region (USEPA, 2003).

Since 1999, WNV has spread throughout the US and North America, resulting in the deaths of more than 450 people by the end of 2003 (Marra et al., 2004). In 2003, the Center for Disease Controls and Prevention (CDC) Division of Vector-Borne Infectious Diseases recorded 9,862 cases of WNV causing 264 deaths in 46 states (CDC, 2004). During the same time, scientists have also observed high rates of avian mortality, composed mostly of American crows (*Corvus brachyrhynchos*) and other corvids, resulting in tens of thousands of recorded deaths per year (Marra et al., 2004). As a result of these impacts on human and non-human species, research and monitoring efforts for WNV in North America have intensified significantly over the past five years.

The transmission cycle of WNV requires mosquitoes, the vectors for the disease. At least 40 species of mosquitoes have tested positive for the virus in North America (Cornell, 2004). The major species associated with the spread of WNV belong to the *Culex* species; cases are linked to *Cx. pipiens*, *Cx. quinquefasciatus*, and *Cx. tarsalis* (CDC, 2003). Mosquitoes feed seeking essential nutrients for egg production. If an infected host is fed on, the mosquito can become a carrier for the virus, and can transfer the virus when it feeds again. Birds tend to develop measurable virus levels (viremia) shortly after being bitten by infected mosquitoes; therefore, they possess the ability to pass the virus onto other mosquitoes if bitten again. Such species are known as “reservoir hosts” because they can pass the virus back to mosquito vectors. Mosquitoes may also infect other animals, including mammals, which are classified as “dead end” hosts, because they do not support a high enough viremia level to successfully pass the virus back to mosquitoes when bitten.

Due to the connections between WNV, mosquitoes, and human and non-human hosts, it is of the utmost importance to understanding the ecology of the virus when making public health decisions. Non-human animal species play a large role in the transmission cycle of the virus. To date, WNV has affected hundreds of species of birds, several species of mammals, reptiles, and a number of domestic animals (USGS, 2004). Some of these species are federally endangered or threatened, while others are ecologically important species. Thus, impacts to these species need to be considered when evaluating the overall impact of mosquito-borne disease. The purpose of this report is to establish a baseline of information regarding the effects of WNV on non-human vertebrate species and to estimate some of the indirect impacts on both human and non-human species as a result.

2. Effects of WNV on Non-Human Species

The regional extent of WNV in North America indicates that a large range of habitats and species are involved in the transmission cycle of the disease. Birds are thought to be key to the spread of the disease. Birds have been shown to be necessary for the transmission of other arboviruses, and have been shown to maintain high levels of WNV in their blood. Mosquitoes frequently feed on birds and, therefore, WNV can be transmitted from birds to mosquito vectors.

In addition, birds suffer from WNV infections. While data on avian deaths in North America are limited, it is estimated that tens to hundreds of thousands of bird deaths have resulted from WNV since its introduction in 1999 (Cornell, 2004). Some species of birds, such as crows and other corvids, appear to be highly susceptible to infection and are more likely to be impacted by WNV. Because of the high infection rate and general visibility of crows, they are often used as an indicator species for West Nile epidemics (Marra et al., 2004).

A variety of mammals, reptiles, and domestic animals are also experiencing the effects of WNV in North America. Horses, after birds, have had some of the highest rates of infection and mortality since 1999. In 2002, 14,358 equine infection cases were recorded in the US, with mortality rates of approximately 30 percent (Cornell, 2004). In addition to horses, other mammals have also experienced the effects of WNV. In total, CDC reported infections in 29 species of mammals as of 2004, including:

- eastern chipmunks
- skunks
- squirrels
- wolves
- sheep
- goats
- bats

- seals
- domestic cats and dogs.

Alligators are another group of large animals that may become infected with WNV (Marra et al., 2004).

The impacts of WNV on the aforementioned species are widespread and can have far-reaching ecological implications. Because WNV is an exotic, non-indigenous virus, its disruption of habitats and population dynamics could potentially be significant. For species such as the greater sage-grouse who already have small populations, the impacts of the virus may lead to the extinction of local and, eventually, regional populations. Furthermore, the migratory patterns of many species, especially birds, may lead to the increased dispersion of the virus over time. Finally, the indirect impacts of population change and ecological damage could impact humans in several ways, including loss of natural controls for diseases and pests and economic impacts from damage to other natural resources.

2.1 Birds

Birds appear to be closely tied to mosquitoes in the transmission cycle of WNV. Birds are thought to act as reservoir hosts for the virus and, subsequently, are associated with the rapid dispersion of the disease. Birds, once infected, can travel for thousands of miles during fall and spring migrations, potentially carrying the virus across the continent or into Central or South America. However, the bird species most impacted by WNV, corvids, generally do not migrate. Corvids, especially crows, tend to be year-round denizens of urban and suburban temperate landscapes, although some species occupy other environments.

WNV has the potential to affect avian species throughout North America. In the short time since the introduction of the virus to the continent, over a hundred species of bird have tested positive for the virus. The total number of species affected and mortalities are likely underestimated due to the limited bird observation network available for monitoring the impacts of the virus. Only a small portion of dead birds are actually sampled for WNV and most dead birds are never observed by people, meaning the potential impact of the disease remains unclear.

2.1.1 Corvids

The outbreak of WNV in 1999 in New York impacted local corvid populations significantly. Corvids highly susceptible to WNV include crows, blue jays, grackles and ravens. The American crow (*Corvus brachyrhynchos*), due to its susceptibility to the virus, has been generally used for surveillance of WNV. The presence, and relative numbers, of dead crows are used to monitor activity of the virus across the nation (Yaremych et al., 2003). The high rate of corvid mortality signals that the virus has a more significant impact on the species than other avian species (Yaremych et al., 2004). However, assessments of the relative impact on corvids may be biased because of the larger size of these birds, the ease of identifying them, their prominent position in suburban and urban ecosystems, and the widespread public knowledge that corvids die from West Nile infection.

In 2000, a total of 1,263 dead crows were confirmed as virus-positive, from 61 of 62 New York State counties (Eidson et al., 2001). Overall, 71,332 avian mortalities were reported to New York State, of which 17,571 (24.6 percent) were crows. For the US as a whole, over 4,000 bird carcasses tested positive for WNV in 2000 (Turell et al., 2003).

Between 1999 and 2003, the New York State Department of Environmental Conservation (NYSDEC) Wildlife Pathology Unit examined over 12,500 avian specimens representing 213 bird species (Chu et al., 2003). Of the 5,950 wild American crows tested, 44 percent were diagnosed with WNV. In addition, 34 percent of dead fish crows (N = 116) and 29 percent of blue jays (N = 1,284) tested positive for the virus during the same time period.

Laboratory experiments with American crows have shown 100 percent death rates from WNV infections. These studies have led researchers to extrapolate that infections in endangered corvid species such as the Hawaiian crow (*Corvus hawaiiensis*), the Mariana crow (*Corvus kubaryi*), and the Florida scrub jay (*Aphelocoma coerulescens*) could lead to significant declines of populations (Yaremych et al., 2004).

2.1.2 Raptors

Birds of prey in the US have been also impacted by WNV. One of the earliest detections of WNV in raptors occurred February 6, 2000 in Westchester County, New York, when a dead red-

tailed hawk tested positive for the virus. Mosquito bites are the most likely mode of infection, but the ingestion of infected reservoir hosts is another possible avenue (Garmendia et al., 2000).

In New York State, 12 percent of tested dead wild hawks tested positive for WNV. In addition, 33 percent of both merlins (N = 15) and American kestrels (N = 33), and 14 percent of great horned owls (N = 63) tested positive for the virus. However, no peregrine falcons (N = 18), bald eagles (N = 25), ospreys (N = 13), barred owls (N = 24), and Eastern screech owls (N = 37) showed any signs of WNV. Captive birds of prey have shown high rates of mortality as evidenced by reports by the US Geological Survey (USGS) National Wildlife Health Center, where nine of 30 tested dead raptors from rehabilitation projects in several states tested positive for WNV (Chu et al., 2003).

2.1.3 Songbirds

Some songbirds other than corvids can be infected by WNV. The NYSDEC Wildlife Pathology Unit found that between 1999 and 2003, 23 percent of tested house finches (N = 43) and 14 percent of tested house sparrows (N = 427) were positive for WNV (Chu et al., 2003). However, aside from corvids such as American crows, fish crows, and blue jays, and the house finches and house sparrows, only three percent of tested dead songbirds tested positive for WNV.

House sparrows may be an important reservoir host for WNV. Studies have shown that when compared with common birds of the northeastern United States, the house sparrow developed the highest levels of viremias and carried the virus for the longest duration of time, up to a maximum of five days (Komar, 2000).

2.1.4 Storks

White storks (*Ciconia ciconia*) that migrate to the Middle East each year from Europe have been identified as susceptible carriers of WNV. In a flock of 1,200 birds found in southern Israel, 13 dead or dying storks were recovered and tested for infection with four showing evidence of WNV in their blood. Six days after the initial outbreak, three of 11 tested birds were seropositive for WNV. In addition, two of 20 white-eyed gulls (*Larus leucophthalmus*) housed in a small pen at the University of Tel-Aviv Department of Zoology were found dead along with several others paralyzed (Malkinson et al., 2002). One hypothesis for the entry of WNV into the

US was that storks shipped to the Bronx Zoo, New York, in 1999 from Israel might have harbored the virus. Deaths in bird populations at the zoo that summer which were not initially attributed to WNV were, in retrospect, among the first signs of the disease.

2.1.5 Sage-Grouse

The greater sage-grouse (*Centrocercus urophasianus*), an endangered species in Canada and under consideration for Federal status in the US, has been impacted by WNV. In four radio-marked populations in the western US and Canada, late summer survival was reduced by 25 percent from infection of WNV. In addition, serum taken from 112 sage-grouses after the outbreak showed that none had antibodies to the virus, suggesting that they lack resistance. The small populations of greater sage-grouse makes WNV a threat to cause its extinction (Naugle et al., 2004).

2.1.6 Domestic Birds

Domestic birds, including chickens and turkeys, have been the subject of several WNV studies due to the potential transmission of WNV to humans by eating infected meat. Also, domestic birds are easy to draw blood from, and so have been used in surveillance for bird-transmitted arboviral encephalitides (Langevin et al., 2001).

Research has shown that WNV is not a likely threat to domesticated birds. Eleven of 12 chickens inoculated with WNV during experiments showed few, if any, clinical signs of the virus (Senne et al., 2000). In addition, Senne et al. observed no transmission of WNV from chicken to chicken under conditions of intimate contact, including exposure to contaminated feces. Langevin et al. (2001) inoculated 21 chickens, none of which developed the clinical disease, confirming Senne et al. Langevin et al. concluded that flock mates and human handlers are not at risk for WNV infection from chickens.

Due to the chicken's ability to seroconvert without developing clinical symptoms, it is often used as a sentinel species for WNV (Langevin et al., 2001; Cornell, 2004). Currently, several states including New York, New Jersey, Maryland, Delaware, Pennsylvania, Florida, Louisiana, and California use chicken flocks for surveillance (CDC, 2003). However, it should be noted that there are differing opinions on the ability of chickens to re-infect mosquitoes with WNV.

Langevin et al. (2001) deemed the magnitude of viremias in chickens insufficient to infect vector mosquitoes, while Senne et al. (2000) suggested that chickens could perpetuate the disease. In addition, sentinel chickens in Suffolk County did not seroconvert although located in areas of high virus activity (McCarthy et al., 2001).

Turkeys are similarly viewed as a potentially important reservoir for WNV due to high density farming techniques. As with chickens, turkeys do not appear to be susceptible to clinical symptoms of WNV when exposed to the disease. Only one of eight inoculated turkeys died after eight days. Control turkeys exposed to the inoculated turkeys showed no WNV-specific antibodies (Swayne et al., 2000). However, CDC testing of turkeys in Wisconsin associated with an outbreak of WNV infections in turkey farm workers found substantial antibody presence (Glaser et al., 2003).

The role of domestic geese (*Anser anser domesticus*) as a WNV reservoir has been supported by observations in Israel and the West Nile region. Infection rates of geese in the Sindbis District of the northern Nile Valley were 27 percent, similar to the rates of buffed-back herons (*Bubulcus ibis ibis*), doves (*Streptopelia senegalensis senegalensis*), and domesticated pigeons (*Columbia livia*), and twice the rate of domesticated chickens and ducks (*Anas platyrhynchos*) (Swayne et al., 2001).

In North America, domestic geese infected with WNV have been found in Manitoba, Canada, where 692 of 2,731 goslings died within a ten-day period at Manitoba Agriculture and Food in Winnipeg (Austin et al., 2004). In addition, a US experiment with goslings resulted in three of four exposed subjects showing signs of depression, weight loss, neck problems including torticollis and opisthotonus, and, eventually, death (Swayne et al., 2001).

2.2 Mammals

WNV has the potential to have ecological effects on non-human mammals. Several species of mammal have tested positive for the virus with multiple deaths observed. Most mammals are considered dead-end hosts, whereby the infected host species are unable to pass the disease back to mosquitoes, and so are not considered part of the transmission cycle. In addition, the majority of mammals are able to produce resistance to the virus; consequently mammal deaths in

previously exposed populations from WNV are rare. One notable exception is horses, which have experienced significant mortality rates.

The majority of mammalian research has focused on horse infections and deaths. Impacts from WNV to non-human primates, and domestic dogs and cats have been documented. In rodents, WNV has been identified in several gray squirrels in Illinois and reports of mass die-offs in the gray squirrel populations were observed in Wyoming (Noler, 2002). Wild raccoons and bats have also seen significant infection and mortality from WNV. Captive species affected include wolves, reindeer, and camels (Graham, 2003).

2.2.1 Horses

Equine cases of WNV have been widespread throughout North America. WNV causes acute, fatal neurological disease at a very high rate, but clinical disease often does not occur in horses (Trock et al., 2001). In 2003, death resulted in approximately 30 percent of the 13,500 WNV infection cases in 39 states (Cornell, 2004). In 2000, 60 horses from seven states met the criteria for confirmed cases; while in 1999, 25 equine cases of WNV were recorded, nine (36 percent) of which resulted in mortality (Ostlund et al., 2001). Twenty-three and 20 cases of equine WNV were recorded in New York State in 2000 and 1999, respectively (Trock et al., 2001).

Experimental studies on equine WNV have shown that horses are not likely to be an amplifying host of the disease. Horses infected with WNV most often develop viremias of low magnitude and short duration that are not conducive to re-infecting mosquitoes or other animal species (Bunning et al., 2002). However, it has been suggested that wild horses may be susceptible to WNV and further research is needed to determine their role in the transmission of the virus (Graham, 2003). Vaccines are currently available for horses in the United States and have shown to be very effective. Frequent injections are required initially, over three to six week intervals, with annual boosters (Cornell, 2004).

2.2.2 Household Pets

Dogs, cats, and other domestic animals have been monitored for WNV due to their close physical relationship with humans. In 1999, testing on 189 dogs in New York City and Nassau County, Long Island, revealed that 5.3 percent had WNV antibodies. None of 12 cats tested had

antibodies for WNV. Dogs are most likely considered dead-end hosts for WNV (Komar et al., 2001). In 2002, an eight-year-old Irish Setter-Golden retriever mix and a three-month-old female wolf pup both died from WNV (Lichtensteiger et al., 2003). It is highly probable that both of these canids were infected by mosquito bites.

An experimental study with dogs suggested they do not play an important role in the epidemiology of WNV. Two of three dogs inoculated with WNV experienced mild clinical illness, but none of the test subjects developed severe disease, and all tested positive for antibodies to the virus (Blackburn et al., 1989).

2.2.3 Rodents

Rodents are often carriers of disease in urban areas. They also are commonly used in scientific research as surrogates for humans. Several experiments involving WNV and rodents have been performed. Most studies reveal that rodents are highly susceptible to WNV, with most test subjects developing severe symptoms and mortalities occurring at a high rate (Mashimo et al., 2002; Xiao et al., 2001). However, it has also been suggested, based on limited data, that wild-bred mice may have the ability to develop complete resistance to the virus (Xiao et al., 2001). Squirrels may also be susceptible to the disease, as studies in Illinois appear to show impacts to several species (Noler, 2002). Additional rodents affected by WNV in the United States include the eastern chipmunk and the black-tailed prairie dog (Marra et al., 2004).

2.2.4 Non-Human Primates

Non-human primates in captive settings in North America have studied for WNV impacts. Due to the close genetic relationship between humans and other primates, evidence of infection has been sought in some situations. In the summer of 2002, following an outbreak of WNV in Louisiana, blood samples were taken from 1,692 captive Rhesus monkeys (*Macaca mulatto*), pigtail macaques (*M. nemestrina*), and baboons (*Papio* spp.) housed outdoors at a primate breeding facility. Overall, 36 percent of the captive non-human primates had antibodies for WNV (Ratterree et al., 2003). However, none of the primates showed clinical illness or neurological disease associated with WNV.

Also during 2002, WNV was observed in an aged Barbary macaque (*Macaca sylvanus*) at the Toronto Zoo (Olberg et al., 2004). However, 30 of 33 other primates at the zoo tested negative for WNV antibodies, including all ten of the remaining Barbary macaques. Three other primates did test positive for antibodies: an olive baboon (*Papio cynocephalus anubis*) and two Japanese macaques (*Macaca fuscata*) (Olberg et al., 2004).

2.2.5 Other Mammals

A variety of other mammals have been found to harbor WNV since 1999. In Illinois, a three-month-old female captive wolf (*Canis lupus*) pup died after being infected with WNV (Lichtensteiger, 2003). In northwest New Jersey, three of 51 black bears tested positive in samples taken in February and March of 2002 (Cornell, 2004). During the same study in New Jersey, seven of 689 white-tailed deer were found to be seropositive for WNV. Other confirmed mammalian deaths in North America from WNV include:

- skunk
- raccoon
- snow leopard
- mountain goat
- llama
- red panda
- rhinoceros
- domestic cattle

(CDC, 2004).

Several documented cases of marine mammals testing positive for WNV have been recorded in recent years. A 12-year-old harbor seal at the New Jersey State Aquarium died as a result of WNV infection in 2002, and two harbor seals from New Mexico's Rio Grande Zoo died from

WNV in 2003. Two populations of Florida manatees, 27 captive individuals, and 108 wild individuals, were tested in 2003. In the captive population, one tested positive for the virus with five others having strong indications of previous infections. All wild manatees tested negative for the virus (Keller et al., 2004).

2.3 Reptiles

WNV has been identified in alligators and crocodiles in the southeastern US. For the most part, WNV affects warm blooded animals – endotherms. For ectotherms, such as alligators and crocodiles, WNV infections are rare. The reports of the disease have all been associated with captive and farmed individuals.

During the fall of 2001 and 2002, captive alligators in southern Georgia experienced WNV epidemics. On one farm, 1,250 out of 10,000 alligators died as a result of WNV infection. The alligators were raised in temperature-controlled buildings and fed a diet of horsemeat supplemented with vitamins and minerals. Due to the relatively low exposure these reptiles have to mosquitoes, it has been hypothesized that those that contracted WNV did so by ingesting infected horsemeat (Miller et al. 2003). Alligators in Florida, Louisiana, and Idaho have also been impacted by WNV. In some cases, it appears that mosquito bites may have been the source of the infection (Wahlberg, 2003). Captive crocodiles (*Crocodylus niloticus*) from multiple farming operations in Israel exhibited high rates of WNV infection (Steinman et al., 2003). These studies indicate a potential for reptilian species to act as a reservoir host for West Nile (Wahlberg, 2003).

3. Potential Ecological Impact

The effects of WNV can extend beyond impacts to specific species and into the ecological niches they inhabit. WNV had been a localized disease found in areas of Africa, Asia, Europe, and the Middle East (Hubalek and Halouzka, 1999). Over the past few years, it has extended throughout North America, and into South America and several Caribbean island nations.

The ecological impacts of WNV are amplified by the rate at which it migrates across continents. Evidence strongly suggests that migratory birds spread the disease (Rappole et al., 2000; Rappole and Hubalek, 2003; Cornell, 2004). Birds acting as reservoir hosts account for the rapid transit of WNV and general patterns of expansion of the disease across the North American continent and into Central and South America (Blitvich et al., 2004). The presence of multiple loci of equine WNV in Central America support this hypothesis (Estrada-Franco et al., 2003; Blitvich et al., 2003). Guadeloupe, in the Caribbean, has also had evidence of West Nile infection in recent years, and this might occur if migratory birds were the reservoir hosts (Quirin et al., 2004).

Certain species react differently to WNV infections. For crows, WNV appears to have a localized impact on populations. Preliminary studies by Cornell Lab of Ornithology researchers showed that at the same time some crow populations in New York City dropped by as much as 90 percent, while nearby crow populations in Long Island showed no significant decrease in population (Chu et al., 2003). A lack of objective, standardized, baseline data makes many conclusions difficult to reach, however, as there are few relevant surveys of the most affected species.

3.1 Impacts on Endangered Species

Endangered and threatened species may be seriously affected by the introduction of WNV. Several avian species have already been identified as being adversely affected by WNV. Chu et al. (2003) noted that the threat of WNV to endangered species such as the Florida scrub jay, California condor, and whooping crane could hamper species recovery plans. The endangered monk seal (*Monachus schauinslandi*) is one of the marine mammals to suffer mortalities from

WNV (Keller, 2004). In general, any populations limited by genetic or geographic barriers will be more susceptible to catastrophic effects, if the species proves to be susceptible to the virus.

3.2 Ecological Niches

The variety of ecological niches in the United States and North American continent make it difficult to predict specific habitat areas that will be affected by outbreaks of WNV. However, effects from the virus have been well documented in certain urban areas. This may be due to high species densities, such as that for crows, or may be due to more relevant monitoring efforts in these regions. Ecological niches most likely to have WNV outbreaks include those near to areas where *Culex* species of mosquitoes breed, or in association with roosting areas of reservoir species of birds.

Anticipation of the arrival of WNV in California led to predictions that the regions most at risk for ecological impacts were the Central Valley, coastal regions, western Sierra Nevada, the Salton Sea, and the lower Colorado basin. These predictions were based on a model that examined the expected impacts of WNV epidemics on wildlife using mosquito vector abundance and the occurrence of sensitive species as variables, while emphasizing the effects on populations over those on individual animal species (Boyce et al., 2004).

However, to date, no specific impacts to particular niches or defined threats to endangered species have been documented in North America.

4. Potential Impact on Humans

The effects of WNV on humans have been well documented in the scientific literature (CDC, 2004; Cornell, 2004; O'Leary et al., 2002). Most humans contract WNV after being bitten by an infected mosquito. The impact of the disease on most people is simply to produce antibodies and show no signs of illness. However, a small percentage of the population will show signs of the disease; sometimes, death can be a result of West Nile infection.

4.1 West Nile Incidence

WNV was first identified in the New York City region in 1999. The strain isolated from the first infections show that the virus was most likely from a human or non-human international traveler from Israel. Since 1999, West Nile has expanded its range from the New York metropolitan area outward in all directions. The rapid geographic expansion of the disease seems to indicate that migratory birds are reservoir species. Others have suggested that interstate transportation serves to convey infected mosquitoes, or infected meat carries the disease from one area to another. These are generally thought to be much less likely causes of the WNV spread. Generally, disease impacts to people are linked to disease incidence in birds (Rappole and Hubalek, 2003).

4.2 Transmission Via Farm Animals

While the major route of human infection is through mosquito bite, some other sources of infection have been identified. In 2002, workers at a commercial turkey breeding farm tested positive for the virus (Glaser et al., 2003). It has been suggested that these workers were infected by some form of non-mosquito transmission. The farming of alligators could also be seen as an area of risk for non-mosquito transmission, as they have been sites of unusual WNV outbreaks in the southeastern United States (Miller et al., 2003; Wahlberg, 2003). No links to these outbreaks and human disease occurrence have been found.

4.3 Consumption of Infected Meat

While no instances of WNV in humans have been attributed to consumption of infected meat, it has been suggested that farmed alligators may have become infected with WNV by eating West Nile-positive horsemeat (Miller et al., 2003). Alligators, with their thick hides, are generally

thought not to be at risk for mosquito bites, and so the consumption of infected horse meat may have been the source of infection. If this is the case, there is the potential that humans could be infected by ingestion of some kind of diseased meat. It has been noted that chickens seroconvert, although they do not appear to become infected with the disease. Turkeys may catch the disease under some conditions, and geese appear susceptible. No data were available for ducks and other widely consumed birds.

5. Indirect Impacts

The indirect impacts of WNV include negative impacts, such as the loss of natural controls and adverse economic impacts. However, there are other impacts that may be viewed as positive, in a sense, such as the decimation of species such as crows, raccoons, and geese that have been identified as pests under some conditions. The indirect impacts of WNV on human and non-human species are, for the most part, undocumented in the scientific literature.

5.1 Loss of Natural Controls of Diseases/Pests

WNV has the potential to impact natural controls of diseases and pests, which may result in effects to agriculture, other human enterprises, or general ecological states. It is undocumented in the scientific literature whether or not West Nile has or will positively or negatively impact these controls. None of the species that appear to suffer during WNV outbreaks are considered to be extremely beneficial to agriculture, for example. In fact, crows have a long history of being pests for many forms of agriculture. None of the impacted species are known to be ecological keystone species, either. The few endangered species impacted by WNV to date have been discussed above. If WNV does indeed have a major impact on raptors, that could lead to ecological shifts, as raptors often fill top of the food chain positions. The importance of top-down controls on food chains has long been the subject of intense ecological study and discussion. Forecasts of any potential impacts would only be reasonable for specific instances in specific settings, however, and is beyond the scope of this discussion.

5.2 Economic Impact

There are many direct economic impacts from WNV. Millions of dollars have already been invested in researching the disease, its transmission cycles, and its impacts on human and non-human species. Increased monitoring efforts will continue to cost millions of dollars, as will preventative measures to control various mosquito species.

The commercial animal industry, including turkey and alligator farming, is likely to see higher costs due to increased monitoring efforts, having to discard diseased animals, and for vaccinations as they become available. For domestic animals, especially horses, the costs will be

similar to that of commercial animals. WNV vaccinations are already widely available for horses, although multiple shots are required in the first year and annual boosters are also required. Of the 20,000 horses vaccinated in Florida during 2001, only one developed WNV symptoms (Cornell, 2004). Costs of the vaccinations are estimated to be \$50 per horse; in 2002, the states of Colorado and Nebraska spent a combined \$2.75 million to vaccinate at-risk horses (Geiser, 2003).

References

- Austin, RJ, TL Whiting, RA Anderson, and MA Drebot. 2004. An outbreak of West Nile Virus-associated disease in domestic geese (*Anser anser domesticus*) upon initial introduction to a geographic region, with evidence of bird to bird transmission. *Canadian Veterinary Journal* 45(2):117-123.
- Boyce, W., C. Kreuder, R. Anderson, and C. Barker. 2004. *Potential Impacts of West Nile Virus on Wildlife in California*. August 26, 2004. <http://www.vetmed.ucdavis.edu/whc/wnv.cfm>.
- Blackburn, NK, F. Reyers, WL Berry, and AJ Shepherd. 1989. Susceptibility of dogs to West Nile Virus: a survey and pathogenicity trial. *Journal of Comparative Pathology* 100:59-66
- Blitvich, BJ, I. Fernandez-Salas, JF Contreras-Cordero, MA Lorono-Pino, NL Marlenee, FJ Diaz, JI Gonzalez-Rolas, N. Obregon-Martinez, JA Chiu-Garcia, WC Black IV, and BJ Beaty. 2004. Phylogenetic analysis of West Nile Virus, Nuevo Leon State, Mexico. *Emerging Infectious Diseases* 10(7):1314-1317.
- Blitvich, BJ, I Fernandez-Salas, JF Contreras-Cordero, NL Marleneem, JI Gonzalez-Rolas, N. Komar, DJ Gubler, CH Calisher, and BJ Beaty. 2003. Serological evidence of West Nile Virus infection in horses, Coahuila State, Mexico. *Emerging Infectious Diseases* 9(7):853-856.
- Bunning, ML, RA Bowen, CB Cropp, KG Sullivan, BS Davis, N. Komar, MS Godsey, D. Baker, DL Hettler, DA Holmes, BJ Biggerstaff, CJ Mitchell. 2002. Experimental infection of horses with West Nile Virus. *Emerging Infectious Diseases* 8(4):380-386.
- CDC. 2004. *West Nile Virus*. August 16, 2004. http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount03_detailed.htm
- CDC. 2003. *Epidemic/Epizootic West Nile Virus in the United States: Guidelines for Surveillance, Prevention, and Control*. 3rd Revision. Centers for Disease Control and Prevention, Fort Collins, CO. 75 pp.
- Chu, M., W. Stone, KJ McGowan, AA Dhondt, WM Hockachka, and JE Therrien. 2003. West Nile File. *Birdscape* Winter:10-11.
- Cornell. 2004. Cornell University Department of Communication and Center for the Environment, Environmental Risk Analysis Program. *West Nile Virus, Transmission, Infections, and Symptoms*. Reviewed August 16, 2004. <http://environmentalrisk.cornell.edu/WNV/Summary2.php>
- Eidson, M., J. Miller, L. Kramer, B. Cherry, Y. Hagiwara, and the West Nile virus Bird Mortality Analysis Group. 2001. Dead crow densities and human cases of West Nile Virus, New York State, 2000. *Emerging Infectious Diseases* 7(4):662-664.
- Estrada-Franco, JG, R. Navarro-Lopez, DWC Beasley, L. Coffey, AS Carrara, AT da Rosa, T. Clements, E. Wang, GV Ludwig, AC Cortes, PP Ramirez, RB Tesh, ADT Barrett, and SC

- Weaver. 2003. West Nile Virus in Mexico: evidence of widespread circulation since July 2002. *Emerging Infectious Diseases* 9(12):1604-1607.
- Garmendia, AE, HJ Van Kruiningen, RA French, JF Anderson, TG Andreadis, A. Kumar, and AB West. 2000. Recovery and identification of West Nile Virus from a hawk in winter. *Journal of Clinical Microbiology* 38(8)::3110-3111.
- Geiser, S., A. Seitzinger, P. Salazar, J. Traub-Dargatz, P. Morley, M. Salman, D. Wilmot, D. Steffen, and W. Cunningham. 2003. *Economic Impact of West Nile Virus on the Colorado and Nebraska Equine Industries:2002*. United States Department of Agriculture, Animal and Plant Health Inspection Services. 4pp.
- Glaser, LC, MV Wegner, JP Davis, ML Bunning, AA Marfin, GL Campbell, B. Bernard, SW Lenhart, and NJ Sotir. 2003. West Nile Virus infection among turkey breeder farm workers – Wisconsin, 2002. *Morbidity and Mortality Weekly Report* 52(42):1017-1019.
- Graham, J. 2003. Animals sick, dying in greater numbers from West Nile Virus. *Chicago Tribune*. September 15, 2003.
- Hubalek, Z and J Halouzka. 1999. West Nile Fever – a reemerging mosquito-borne viral disease in Europe. *Emerging Infectious Diseases* 5(5):643-650.
- Keller, M., MT Long, K. Conley, R. Francis-Floyd, R Isaza. 2004. A serosurvey of marine mammals for West Nile Virus and the development of a competitive inhibition ELISA (Enzym-linked immunosorbent assay). *Presentation abstract from Phi Zeta Research Emphasis Day*. July 14, 2004. <http://www.vetmed.ufl.edu/phizeta/phizetasched04.htm>
- Komar, N., NA Panella, and E. Boyce. 2001. Exposure of domestic mammals to West Nile Virus during an outbreak of human encephalitis, New York City, 1999. *Emerging Infectious Diseases* 7(4):736-738.
- Komar, N. 2000. *House Sparrows as Reservoirs of West Nile*. Paper presented at American Journal of Tropical Medicine and Hygiene Conference. November 1, 2000. Scientific Session S, Paper 226.
- Langevin, SA, M. Bunning, B. Davis, and N. Komar. 2001. Experimental infection of chickens as candidate sentinels for West Nile Virus. *Emerging Infectious Diseases* 7(4): 726-729.
- Lichtensteiger, CA, KH Taheny, TS Osborne, RJ Novak, BA Lewis, and ML Firth. 2003. West Nile Virus encephalitis and myocarditis in wolf and dog. *Emerging Infectious Diseases* 9(10):1303-1306.
- Malkinson, M., C. Banet, Y. Weisman, S. Pokamunski, R. King, MT Drouet, and V. Deubel. 2002. Introduction of West Nile Virus in the Middle East by migrating white storks. *Emerging Infectious Diseases* 8(4):392-397.
- Marra, PP, S. Griffing, C. Caffrey, AM Kilpatrick, R. McLean, C. Brand, E. Saito, AP Dupuis, L. Kramer, and R. Novak. 2004. West Nile Virus and wildlife. *BioScience* 54(5):393-402.

- Mashimo, T., M. Lucas, DS Chazottes, MO Frenkiel, X. Montagutelli, PE Ceccaldi, V. Deubel, JL Guenet, and P. Despres. 2002. A nonsense mutation in the gene encoding 2'-5'-oligoadenylate synthetase/L1 isoform is associated with West Nile Virus susceptibility in laboratory mice. *Proceedings of the National Academy of Sciences* 99(17):11311.
- McCarthy, TA, JL Hadler, K. Julian, SJ Walsh, BJ Biggerstaff, SR Hinten, C. Baisley, A. Iton, T. Brennan, RS Nelson, G. Archambault, AA Marfin, and LR Petersen. 2001. West Nile serosurvey and assessment of personal prevention efforts in an area with intense epizootic activity: Connecticut, 2000. pp. 307-316. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.
- Miller, DL, MJ Mauel, C. Baldwin, G. Burtle, D. Ingram, ME Hines, II, and KS Frazier. 2003. West Nile Virus in farmed alligators. *Emerging Infectious Diseases* 9(7):794-799.
- Naugle DE, CL Aldridge, BL Walker, TE Cornish, BJ Moynahan, MJ Holloran, K. Brown, GD Johnson, ET Schmidtman, RT Mayer, CY Kato, MR Matchett, TJ Christiansen, WE Cook, T. Creekmore, RD Falise, ET Rinkes, and MS Boyce. 2004. West Nile Virus: pending crisis for greater sage-grouse. *Ecology Letters* 7:704-713.
- Noler, RS. 2002. Human, animal cases of West Nile continue to climb. *Journal of the American Veterinary Medical Association* 221(9) News section..
- Olberg RA, IK Barker, GJ Crawshaw, MF Bertelsen, MA Drebot, and M. Andonova. 2004. West Nile virus encephalitis in a Barbary macaque (*Macaca sylvanus*). *Emerging Infectious Diseases* 10(4):712-714.
- O'Leary, DR, AA Marfin, SP Montgomery, AM Kipp, JA Lehman, BJ Biggerstaff, VL Elko, PD Collins, JE Jones, and GL Campbell. 2002. The epidemic of West Nile Virus in the United States, 2002. *Vector-Borne and Zoonotic Diseases* 4(1):61-70.
- Ostlund, EN, RL Crom, DD Pedersen, DJ Johnson, WO Williams, and BJ Schmitt. 2001. Equine West Nile Encephalitis, United States. *Emerging Infectious Diseases* 7(4):665-669.
- Quirin, R., M. Salas, S. Zientara, H. Zeller, J. Labie, S. Murri, T. Lefrancois, M. Petitclerc, and D. Martinez. 2004. West Nile Virus, Guadeloupe. *Emerging Infectious Diseases* 10(4):706-708.
- Rappole, JH, and Z. Hubalek. 2003. Migratory birds and West Nile Virus. *Journal of Applied Microbiology* 94:47S-58S.
- Rappole, JH, SR Derrickson, and Z. Hubalek. 2000. Migratory birds and spread of West Nile Virus in the Western Hemisphere. *Emerging Infectious Diseases* 6(4): 319-328
- Ratterree, MS, APA Travassos da Rosa, RP Bohm, Jr., FB Cogswell, KM Phillippi, K. Caillouet, S. Schwanberger, RE Shope, and RB Tesh. 2003. West Nile Virus infection on nonhuman primate breeding colony, concurrent with human epidemic, southern Louisiana. *Emerging Infectious Diseases* 9(11):1388-1394.

- Senne, DA, C. Pedersen, DL Hutto, WD Taylor, BJ Schmitt, and B. Panigrahy. 2000. Pathogenicity of West Nile Virus in chickens. *Avian Diseases* 44:642-649.
- Steinman, A., C. Banet-Noach, S. Tal, O. Levi, L. Simanov, S. Perk, M. Malkinson, and N. Shpigel. 2003. West Nile Virus in crocodiles. *Emerging Infectious Diseases* 9(7):887-889.
- Swayne, DE, JR Beck, CS Smith, WJ Shieh, and SR Zaki. 2001. Fatal Encephalitis and Myocarditis in young domestic geese (*Anser anser domesticus*) caused by West Nile Virus. *Emerging Infectious Diseases* 7(4):751-753.
- Swayne, DE, JR Beck, and S. Zaki. 2000. Pathogenicity of West Nile Virus for turkeys. *Avian Diseases* 44:932-937.
- Trock, SC, BJ Meade, AL Glaser, EN Ostlund, RS Lancotti, BC Cropp, V. Kulasekera, LD Kramer, and N. Komar. 2001. West Nile Virus outbreak among horses in New York State, 1999 and 2000. *Emerging Infectious Diseases* 7(4):745-747.
- Turell, MJ, M. Bunning, GV Ludwig, B. Ortman, J. Chang, T. Speaker, A. Spielman, R. McLean, N. Komar, R. Gates, T. McNamara, T. Creekmore, L. Farley, and CJ Mitchell. 2003. DNA vaccine for West Nile Virus infection in fish crows (*Corvus ossifragus*). *Emerging Infectious Diseases* 9(9):1077-1081.
- USEPA. 2003. Office of Water. *Wetlands and West Nile Virus*. Brochure. EPA-843-F-03-012.
- USGS. 2004. *Species Affected by West Nile Virus*. August 23, 2004. http://www.nwhc.usgs.gov/research/west_nile/Species%20Affected%20List%202004.doc
- Wahlberg, D. 2003. Alligators can transmit West Nile Virus, outbreak shows. *The Atlanta Journal – Constitution*. November 23, 2003.
- Xiao, SY, H. Guzman, H. Zhang, APA Travassos da Rosa, and RB Tesh. 2001. West Nile Virus infection in the golden hamster (*Mesocricetus auratus*): a model for West Nile Encephalitis. *Emerging Infectious Diseases* 7(4):714-721.
- Yaremych, SA, RE Warner, MT Van de Wyngaerde, AM Ringia, R Lampman, and RJ Novak. 2003. West Nile Virus detection in American crows. *Emerging Infectious Diseases* 9(10):1319-1321.
- Yaremych, SA, RE Warner, PC Mankin, JD Brawn, A Raim, and R Novak. 2004. West Nile Virus and high death rate in American crows. *Emerging Infectious Diseases* 10(4):709-711.